SUMMARY. Observations are recorded on 100 cases of nephritis showing creatinine retention. Of these 100 cases, 85 had a creatinine of over 5 mg. per 100 c.c. of blood, the figures ranging from 5.1 to 33.3 mg.; 80 of these 85 cases have died. Of the 5 remaining the condition of 3 is essentially unchanged, while 2 have recovered. These 2 cases showed only a temporary elevation of the blood creatinine.

A considerable number of the 85 cases were able to be up and about and some showed decided clinical improvement. The creatinine gave us a better prognostic insight into these cases than either the blood urea or phthalein tests, which were made simultaneously. It is our opinion that in advanced cases of nephritis the blood creatinine furnishes a more reliable prognosis than any other test we possess.

REMARKS ON OCULOGYRATION AND ON THE LESION CAUSING COMPLETE BILATERAL OPHTHALMOPLEGIA.[1]

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FRENCH neurologists employ the term *oculogyre* and its various modifications. In seeking English equivalents for these terms I have consulted the *Standard Dictionary*, as I am informed by certain ophthalmologists with whom I have conferred that the French usage is not common in American ophthalmology. A gyre may mean a gyration, it may also mean a gyrus, as one of the convolutions of the brain. Gyration means: "The act of turning, as a body on its axis of rotation, while simultaneously whirling about an axis of revolution," and it seems better adapted to the translation than gyre. I have employed the terms dextrogyration and sinistrogyration to mean right lateral associated movement of the eyeballs and left lateral associated movement, respectively.

The attempt is made in this paper to discuss certain aspects of oculogyration and to distinguish clinically between lesions above and below the nucleus of the branch of the oculomotor nerve supplying the internal rectus muscle, causing disturbance of lateral associated ocular movement; also to explain the lesion causing complete bilateral ophthalmoplegia in a way I have not seen employed in literature. A lesion at a considerable distance above the oculomotor nucleus probably could not cause persistent paralysis of ocular movements, as such movements are bilaterally represented in the cerebral cortex.

G. S., aged fifty-two years, was admitted to the University Hospital December 8, 1918. He used alcohol in moderation. The downward and inward rotation of the right eyeball from the action of the superior oblique was distinct when he came to the hospital, on December 8, 1918. External rotation of the right eyeball was preserved, but there was complete right oculomotor palsy. An examination revealed nothing of importance in addition to the ocular condition. His gait and tendon reflexes were normal. The Wassermann test of the blood was negative; a spinal puncture gave 8 c.c. of fluid; the cell count was 11 cells per cubic millimeter; the pressure of the spinal fluid was slightly elevated; the Wassermann and globulin tests of the fluid were negative.

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[1] Read before the Philadelphia Neurological Society, February 28, 1919.

He brought the following note from Dr. Alfred Cramer: "When I first saw you on November 11, 1918, you told me that your eyes had given you trouble for only two weeks previously. You complained chiefly of seeing double, though your general health had been below par for some time. I doubt if you had influenza at the beginning of your trouble. My examination showed there was a palsy of the superior rectus muscle of the right eye and a partial drooping of the right eyelid. The rotation of the eyeballs was good in other directions. The state of your candle field with a red glass before the left eye showed there was diplopia in the primary position, the red candle showing below and slightly to the left-hand side. As the candle was moved down toward the lower field the candle flames came nearer and nearer together until they apparently fused. Neither flame seemed to be oblique, except in rotation up and to the right.

"The condition remained about the same until November 29, but during the week since this date there has been considerable change. I now find there is a complete paralysis of all the muscles of the right eye, with the exception of the external rectus muscle and superior oblique. There is also inability to lift the right upper eyelid. There also seems to be a slight limitation of the upward rotation of the left eyeball. There is, too, a paralysis of the iris, the pupil being immobile and partially dilated. So far as I can see there are no changes in either eye-ground. The vision in each eye is practically normal without glasses. Negative Wassermann."

Notwithstanding the negative findings the man was treated as a possible syphilitic, and on December 15, seven days after admission to the hospital, some improvement in the oculomotor palsy of the right eye was noticed, and this improvement continued; but it was soon observed that he had no rotation of the right eye to the right of the midline. Dr. J. T. Carpenter confirmed this observation on December 27, and also reported that the eye-grounds showed no specific lesions and both disks showed clear edges and there was no swelling. Soon after this it was observed that the left internal rectus was very weak.

At the present time the complete right oculomotor palsy has changed considerably; he now has some inward and downward movement of the right eye; no ptosis of the upper lid, but complete paralysis of associated upward movement of the eyeballs and nearly complete paralysis of dextrogyration of the eyeballs and partial palsy of sinistrogyration; nor does the left internal rectus function as well in convergence as in dextrogyration. The downward motion of the left eye is normal or nearly normal.

At the time the right oculomotor palsy was complete, Dr. Cramer found, December 6, 1918, a slight limitation of the upward rotation of the left eyeball. The gradual but pronounced improvement of the motion of the right eye was associated with a nearly complete paralysis of outward motion of the right eye and of upward motion of the left eye. It might be thought that this external rectus palsy was produced by the use of arsenobenzol, but the additional palsy of the left internal rectus made the palsy an associated one, which could hardly be caused by this drug.

Paralysis of upward associated movement, as I[2] have shown by previous observations, indicates a lesion near the corpora quadrigemina. There is evidently a lesion in this man in or near the corpora quadrigemina, causing paralysis of upward associated movement, marked paralysis but not complete of dextrogyration and slighter paralysis of sinistrogyration; and yet he presents no incoördination of the limbs, as one might expect, from disorder of the superior cerebellar peduncles. The lesion cannot be large enough to cause implication of these structures.

Were his condition associated with hemiplegia the symptom-complex would be what the French writers call the syndrome of Foville, and such a case was reported recently by André Leri and Perpère.[3]

A case of paralysis of oculogyration on the side opposite to the hemiplegia was reported by Dr. Potts and myself from a tubercle of the pons,[4] and this heterolateral paralysis is indicative of a lesion of the pons; for example, paralysis of dextrogyration with eyes turned to the left associated with left hemiplegia indicates a lesion of the right side of the pons. It means that a lesion of the connecting fibers of the nucleus of the nerve to the internal rectus of one eye with the nucleus of the opposite abducens nerve has occurred, and usually near the abducens nucleus. In this form the function of the affected internal rectus muscle may be preserved in convergence, indicating that the nucleus of the nerve to this internal rectus and its cortical connection cannot be destroyed.

I believe that when the central pathway of corticonuclear oculogyration is paralyzed above or in the nucleus of the nerve to the affected internal rectus the function of this muscle in convergence will disappear or become much impaired, as it did in the case I now report. When the break occurs in these fibers above this nucleus no impulses can pass into this nucleus for either form of function of the internal rectus, *i. e.*, for oculogyration or for convergence; but when the break occurs in the posterior longitudinal bundle, and therefore below the nucleus of the nerve to the internal rectus, the function of the internal rectus muscle in convergence may be preserved, as it has no relation to the abducens nerve, but oculogyration demanding the function of this muscle in association with the function of the heterolateral external rectus will be lost. So far as I know this diagnostic distinction of a lesion above the nucleus of the nerve to the internal rectus as compared with one in the posterior longitudinal bundle below this nucleus as of value in localization, has not been described, and it applies to the lesion above the nucleus only when it is near the nucleus.

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[2] Jour. Nerv. and Ment. Dis., 1905, and Obersteiner's Arbeiten, vols. xv and xvi.

[3] Rev. Neur., May–June, 1918, 530.

[4] Univ. of Penna. Med. Bull., December, 1903.

As the patient under discussion has paralysis of upward associated movement with paralysis of dextrogyration and paresis of sinistrogyration, and still less power of the left internal rectus in convergence than in the associated movement of this muscle with the right external rectus, he probably has a lesion of the corpora quadrigemina, and not one on the base involving the nerves at their exit.

It is not necessary to assume that the paralysis of the right external rectus muscle in this case is caused by a lesion of the abducens nerve or its nucleus. Complete ophthalmoplegia implicating all the nerves supplying the eyeballs has been explained usually as the result of inflammatory lesions of all the ocular nuclei, but this explanation, when all other cranial nerves entirely escape, has seemed to me unsatisfactory, especially as bilateral complete ophthalmoplegia has been repeatedly observed without other nerve lesions. I believe that when a lesion destroys the nuclei of the trochlearis and oculomotorius nerves bilaterally, which a comparatively small lesion may readily do, as these nuclei are very near together, it must destroy the connections in the posterior longitudinal bundles between the nuclei of the internal recti and those of the external recti, and consequently paralysis of the external recti muscles will result without any lesion of the abducens nerves or their nuclei. The movements of the two eyeballs are intimately associated and a lesion of the central pathway of corticonuclear oculogyration near the nuclei affects the movements of both eyeballs.

Dr. T. B. Holloway made the following report, April 16, 1919:

Your patient was seen by me April 15, and I found that since his last observation the vision of his left eye had deteriorated considerably and is now but 3/150. The other conditions were as follows:

The veins of the upper lid of the left eye are more prominent than those of the right. There is a ptosis of the left upper lid, the palpebral fissure measuring 6 mm. in contrast to 10.5 mm. for the fellow eye. The left eye is distinctly proptosed and displaced downward 2.5 mm. and outward 1 mm. Hertel's exophthalmometer shows the apex of the left cornea to be 3.5 mm. more than the right. There is decided resistance to backward displacement of the globe. No visible or palpable pulsation is present. I did not have a stethoscope to listen for a bruit. Palpation of the anterior portion of the orbit reveals a distinct fulness at the upper and inner angle. The caruncle and plica are reddened and prominent.

Upon rotation of the globes to the right there is a decided restriction of movements, the right eye rotating outward and the left inward not more than 10 degrees. Upon rotation to the left the left eye rotates to within 2 mm. of the external canthus, while the rotation inward of the right eye is hardly as great. The upward rotation is restricted in both eyes, but distinctly more so in the left. There is slight downward restriction of both eyes. Upon attempts to converge, the left eye practically fails and soon deviates outward.

The right pupil measures 4.5 mm., is distinctly eccentric and is displaced upward and slightly inward; the position of the left pupil is normal and it measures 3 mm. There is no direct or indirect pupillary light reflex in the right eye, but in the left there is a slight direct and greater indirect light reflex. Both pupils react slightly upon attempts at convergence and accommodation.

The media are clear, the disks are not pathological and there are no fundus changes aside from slight increase of the arterial reflex and some venous pressure signs, due to incipient sclerosis of the vessels.

In the left eye the retinal veins appear to be a trifle fuller and darker than in the right.

The field of the right eye does not show any changes for form or colors, while that of the left shows a cut across the upper portion of the form field, to about 25 degrees, that seems to be more than would be accounted for by the ptosis. With the left eye he could not notice any colors in a 10 mm. circle.